ANALYSIS OF ECG CHANGES AS A MANIFESTATION OF CARDIAC COMPLICATIONS OF ORGANOPHOSPHOROUS COMPOUND POISONING

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DECLARATION

I solemnly declare that this dissertation entitled "ANALYSIS OF ECG CHANGES AS A MANIFESTATION OF CARDIAC COMPLICATIONS OF ORGANOPHOSPHOROUS COMPOUND POISONING" was done by me at Madras Medical College, Government General Hospital, during 2010 under the guidance and supervision of Prof. C. RAJENDIRAN, M.D. This dissertation is submitted to the Tamil Nadu Dr.M.G.R Medical University towards the partial fulfillment of requirements for the award of M.D.Degree in General Medicine(Branch 1)

Place: Chennai-3 candidate's signature.
Date:

CERTIFICATE

This is to certify that the dissertation entitled "ANALYSIS OF ECG CHANGES AS A MANIFESTATON OF CARDIAC COMPLICATIONS OF ORGANOPHOSPHOROUS COMPOUND POISONING" is a bonafide work done by Dr.S.Geetha at Madras Medical College, Chennai in partial fulfillment of the university rules and regulations for award of M.D.,Degree in General Medicine (Branch 1) under my guidance and supervision during the academic year 2010.

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CONTENTS

INTRODUCTION

Organophosphorus compounds are chemical agents in widespread use throughout the world mainly in agriculture. They comprise the ester, amide or thiol derivatives of phosphoric acid and are most commonly used as pesticides in commercial agriculture.

There are no rules and regulations governing the purchase of these products, and they are therefore readily available "over the counter", despite them being a major cause of morbidity and mortality. Poisoning occurs either by a suicidal attempt or by an accidental exposure of the compound during spraying.

Estimates from the WHO indicate that each year, 1 million accidental poisonings and 2 million suicide attempts involving pesticidesoccur world over. Early diagnosis and prompt treatment is required to save the patient's life[1]. The incidence of intentional organophosphate related human exposure is underestimated[1.3]

Large proportion of patients present to the ICU with acute suicidal [15,16] attempt and male predominance.

REVIEW OF LITERATURE

Organophosphates were first synthesised in the early 1800 s. when Lassaigne reacted alcohol with phosphoric acid. A chemist at Bayer AG Germany investigated the use of organophosphates as insecticide during World War II in 1941, Organophosphates were reintroduced world wide as pesticides [45].

Exposure to organophosphates is also possible via intentional or unintentional contamination of food sources. Although no clinical effects of chronic low level exposure from food sources have been shown, advancement in risk assessment and preparedness are on going [46].

Organophosphates may affect children or other at risk populations differently. The increased susceptibility has not been elucidated but may involve delayed or persistant effects. More work in this area is under way [47].

INCIDENCE AND PREVALANCE

Most cases occur in the developing world as a result of occupational or deliberate exposure to organophosphate pesticides. Organophosphates seem to be the most important cause of death from self poisoning world wide. Case mortality rate in the developing world is more than 20% [49].

A retrospective study of organophosphate poisoned patients by Livetal found a direct correlation between severity of poisoning and mortality and the presence of pretreatment metabolic and respiratory acidosis [50].

CLASSIFICATION

There are classified according to their toxicity and clinical use:

1. Highly toxic - (e.g. parathion).
2. Intermediately toxic - (e.g clorpyriphos, trichlorfon)
3. Low toxicity - (e.g. diazinon, malathion, dichlorvos).

MECHANISM OF ACTION OF ORGANOPHOSPHORUS COMPOUNDS

Acetylcholine is the neurotransmitter released at all postganglionic parasympathetic nerve endings and at the synapses of both sympathetic and parasympathetic ganglia. It is also released at the skeletal muscle myoneural junction and serves as the neurotransmitter in the CNS [18]. It is hydrolyzed by acetylcholinesterase into two fragments: acetic acid and choline.

Acetylcholinesterase is present in two forms: True acetylcholinesterase which is found primarily in the tissues and erythrocytes, and pseudocholinesterase which is found in the serum and liver [19].

Accumulation of acetylcholine causes overstimulation of both muscarinic and nicotinic receptors, and subsequently disrupts the transmission of nerve impulses in both the peripheral and central nervous system.

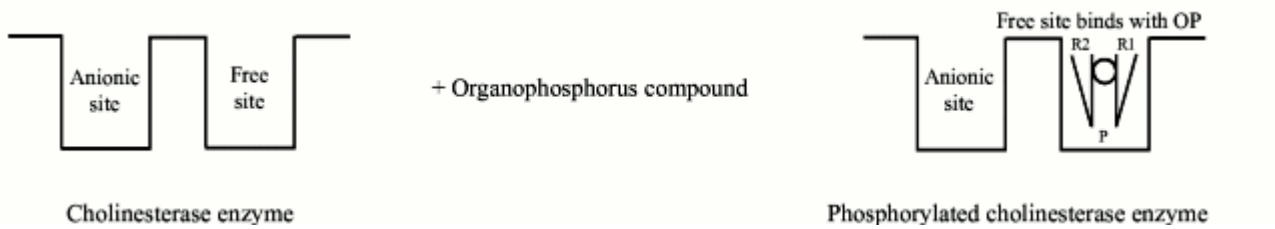
PHARMACOKINETICS

Most organophosphates are highly lipid soluble compounds and are well absorbed from intact skin, oral mucous membranes, conjunctiva and the gastrointestinal and respiratory tracts. They are rapidly redistributed to all body tissues. The highest concentrations are found in the liver and kidneys. As they are highly lipid soluble they easily cross the blood brain barrier and therefore produce potent effects on the central nervous systeml

Metabolism occurs principally by oxidation in the liver by conjugation and esterase hydrolysis producing a half-life of minutes to hours. The oxidative metabolites of malathion and parathion are active forms. Elimination of organophosphorus compounds occur via urine, bile and faeces.

CLINICAL FEATURES OF ORGANOPHOSPHORUS POISONING

Following exposure toxic features are obvious within 30 minutes to 3 hours. They may be delayed in some cases depending on the rate and amount of systemic absorption.



The clinical symptoms and signs are non-specific and depend on the quantity of the specific agent and the route of entry. Most fatalities occur within 24 hours and those who recover do so within 10 days.

The clinical features can be broadly classified as secondary to the

a. Muscurinic effects

b. Nicotinic effects and

c. Central receptor stimulation[5]

TABLE 1 : **SYMPTOMS AND SIGNS OF ORGANOPHOSPHORUS POISONING**

MUSCARINIC RECEPTORS	NICOTINIC RECEPTORS	CENTRAL RECEPTORS
CARDIOVASCULAR o Bradycardia o Hypotension **RESPIRATORY** o Rhinorrhoea o Bronchorrhoea o Bronchospasm o Cough **GASTROINTESTINAL** o Nausea/vomiting o Increased salivation o Abdominal cramps o Diarrhoea	**CARDIOVASCULAR** o Tachycardia o Hypertension **MUSCULOSKELETAL** o Weakness o Fasciculations o Cramps o Paralysis **EYES** o Blurred vision o Miosis o Increased lacrimation	**GENERAL EFFECTS** o Anxiety o Ataxia o Insomnia o Convulsions o Tremors o Coma o Respiratory depression o Circulatory collapse **GLANDS** o Excessive salivation

CARDIAC MANIFESTATIONS

The extent ,frequency and pathogenesis of the cardiac toxicity from these compounds are not clearly defined. The commonest cardiac manifestations are hypotension (with warm, dilated peripheries), and bradycardia. Electrocardiographic manifestations include prolonged Q-Tc intervals, elevation of the ST segment, inverted T waves and a prolonged PR interval.QT prolongation has been found in a higher incidence of respiratory failure and mortality[39].

Three phases of cardiac toxicity following organophosphate poisoning have been described:

- **Phase I**: A brief period of increased sympathetic tone
- **Phase II**: A prolonged period of parasympathetic activity including AV node blockade
- **Phase III**: Q-T prolongation followed by torsade de pointes, ventricular tachycardia and ventricular fibrillation.

However, according to a recent report the mortality rate has declined considerably following intensive management[5].

Although bradycardia is thought to dominate in the early cholinergic phase of the poisoning sinus tachycardia was more frequent in our study. Similar observations have been made by others [16].

The mechanism of cardiac toxicity is unclear and the following have all been postulated:

- A direct toxic effect on the myocardium
- Over activity of cholinergic or nicotinic receptors causing hemodynamic alteration.
- Hypoxia
- Acidosis
- Electrolyte abnormalities
- High dose atropine therapy.

One case of organophosphate – parathion poisoning in a farmer with suicidal attempt developed myocardial infarction and succumbed has been described by Yagneshetal [51].

RESPIRATORY MANIFESTATIONS

Respiratory manifestations of acute organophosphorus poisoning include bronchorrhoea, rhinorrhoea, bronchospasm and laryngeal spasm - due to muscarinic effects. Nicotinic effects lead to weakness and subsequent paralysis of respiratory and oropharyngeal muscles which may require mechanical ventilation. Central neurological depression may lead to respiratory arrest due to cholinergic stimulation. Acute respiratory failure is reported in 33% of patients with acute poisoning [20].

GASTROINTESTINAL SYMPTOMS

Vomiting, diarrhea and abdominal cramps are the first to occur after oral ingestion of an organophosphorus compound .Acute pancreatitis [21,22] has been reported.Patients may have hypo or hyperglycemia.[23]

Acute haemorrhagic pancreatitis has been reported a complication of organophosphate poisoning.

A high index of suspicion has to be there to diagnose the condition early and treat accordingly[52].

NEUROLOGICAL MANIFESTATIONS

Three different types of paralysis are recognized based largely on the time of occurrence and their differing pathophysiology:

- Type I paralysis or acute paralysis
- Type II paralysis or Intermediate syndrome
- Type III paralysis or Organophosphate- induced delayed polyneuropathy

TYPE I PARALYSIS OR ACUTE PARALYSIS

This is seen during the initial cholinergic phase. There is persistent depolarization at the neuromuscular junction. Clinical features include muscle cramps, fasciculations, twitching and weakness. Patients may require ventilatory support due to the weakness of the respiratory muscles leading to respiratory depression and arrest. Acute paralysis responds to atropine[24].

TYPE II PARALYSIS OR INTERMEDIATE SYNDROME

First described in 1974 by Wadia et al[14] as type II paralysis and subsequently termed "The Intermediate Syndrome" by Senanayake. Since then there has been numerous reports of this syndrome. [24,26,27,28,29] The incidence of this syndrome is between 8% and 49%.[28,29,30] This develops 24-96 hours after the poisoning. The cardinal feature of this syndrome is muscle weakness affecting the proximal limb muscles and neck flexors. The cranial nerves supplying the extra-ocular muscles are mostly involved. This phase persist for 4-18 days. Nerve conduction shows axonal neuropathy [31].Studies from CMC Vellore have shown significant elevation of muscle enzymes.[32]

TYPE III PARALYSIS OR ORGANOPHOSPHATE- INDUCED DELAYED POLYNEUROPATHY

Type III paralysis is a sensory motor distal neuropathy that usually occurs after ingestion of large doses of organophosphorus compounds[9-11] This presents as weakness and ataxia following a latent period of 2-4 weeks. Initial stimulation causes excitatory fasciculation, which then progresses to an inhibitory paralysis. The cardinal symptoms are distal weakness of the hands and feet .Neuro psychiatric manifestations have been reported.[33] Delayed CNS signs include tremor, anxiety and coma. Cogwheel tremors,

choreoathetosis [34] gullian barre syndrome, [35]sphincter involvement,[36] bilateral recurrent larymgeal nerve paralysis [37]and ototoxicity [38]has been reported. Recovery occurs in 6-12 months.

DIAGNOSIS

Diagnosis of organophosphorus poisoning requires a high index of suspicion. The history of exposure may be denied by patients who have attempted suicide, or unavailable in unconscious patients. Signs of poisoning include the pungent garlic- like odour of organophosphorus in breath and vomitus, miosis, bradycardia and muscle fasciculations. Some patients may present with the nicotinic effects of tachycardia, hypertension and mydiasis (rather that the anticipated bradycardia and hypotension).

Treatment is initiated immediately, without waiting for blood investigations. Both true and pseudocholinesterase levels have to be estimated to assess poisoning True cholinesterase levels correlate with severity of the poisoning, pseudocholinesterase levels do not [40]. A 25% or more of the depression of the RBC cholinesterase is taken as an indicator of poisoning [19].

The level of cholinesterase activity is relative and based on population estimates. Neonates and infants have baseline levels that are lower than adults. The diagnosis is confirmed by observing a progressive increase in cholinesterase values till the values plateau over time.

Falsely depressed levels of RBC cholinesterase can be found in pernicious anemia, haemoglobinopathies, use of antimalarial drugs and oxalated blood tubes.

MANAGEMENT OF ORGANOPHOSPHORUS COMPOUNDS POISONING

- Skin decontamination
- Airway protection if indicated
- Gastric lavage
- Activated charcoal 0.5-1gm/kg every 4hr
- Anticholinesterase: Atropine/glycopyrrolate
- Cholinesterase reactivator: Pralidoxine
- Ventilatory support
- Inotropic support
- Feeding-enteral/parental

MANAGEMENT

DECONTAMINATION

Patient should be removed from the site of exposure and their clothes removed. Patient's body should then be thoroughly washed with soap and water to prevent further absorption from the skin. Gastric lavage is done. In unconscious patients the airway needs to be protected. Patient should receive activated charcoal 0.5-1 gm/ kg every four hours to reduce absorption in the gastrointestinal tract. Adverse effects of activated charcoal include aspiration pneumonia, vomiting, diarrhea, constipation, ileus and reduced absorption of oral medication [53, 54 & 55]. Lavage is preferred to enforced emesis as this may precipitate seizures. Recent study has shown the addition of serotonin adipinate resulted in shortening of the cholinergic phase and a 3.5% reduction in the mortality [41].

AIRWAY AND RESPIRATION

The airway should be secured and adequate oxygenation ensured. Careful observation of the respiratory status is required as these patients are prone to develop respiratory failure during both the acute phase and during intermediate syndrome. The following should be monitored on a regular basis to assess the patient's respiratory status:

- Respiratory rate

- Tidal volume/ vital capacity
- Neck muscle weakness
- Ocular muscle involvement eg. diplopia
- Arterial blood gas analysis

CARDIAC MONITORING

As mentioned earlier, a wide range of cardiac manifestations can occur and careful haemodynamic and electrocardiac monitoring should be undertaken in all patients. It should be remembered that hypoxaemia, metabolic and electrolyte abnormalities can all contribute to cardiac arrhythmias. Some arrhythmias may require cardiac pacing.

ATROPINE.

Administration of anticholinergics is still the mainstay of treatment, and should be started as soon as the airway is secured. Starting dose of atropine is 2mg IV bolus. Subsequent doses of 2-5mg every 5-15 minutes should be administered until atropinization is achieved. Signs of adequate atropinization include an increased heart rate (>100 beats/min.), moderately dilated pupils, a reduction in bowel sounds, a dry mouth and a decrease in bronchial secretions. The dose of atropine required is maximal on day 1 and tends to decrease over the next few days. Atropine does not reverse the skeletal muscle effects.

Excessive treatment with atropine results in toxicity characterised by confusion and tachycardia [56].

GLYCOPYROLATE

Some studies have shown that glycopyrrolate is equally effective, with fewer central nervous system side effects and a better control of secretions[12].

CHOLINESTERASE REACTIVATOR

Oximes are nucleophilic agents that re-activate the phosphorylated acetylcholinesterase by binding to the organophosphorus molecule. Obidoxime is the most potent agent with some organophosphorus compounds and HI6 against [42] others. Pralidoxime is less potent.[42,43,44]

Pralidoxime has three main actions:

- A direct action converting the organophosphate to a harmless compound.
- A transient reaction protecting the enzyme from further inhibition.
- Reactivation of the inhibited alkyl phosphorylated enzyme to free the active unit (if given early enough)

Pralidoxime should be started as early as possible to prevent permanent binding of the organophosphate to acetylcholinesterase. Once this has occurred, receptor regeneration is required to allow recovery. Pralidoxime should be continued until adequate spontaneous ventilation is achieved by the patient. The effective plasma concentration is 4mg/litre and the patient should show signs of improvement 10- 40 minutes after its administration. Plasma and pseudocholinesterase levels should ideally be monitored during treatment.

PREVENTION

Improved regulation of the availability of pesticides, strict regulation of vendors, and modifications in packaging of pesticides may all help reduce the use of organophosphates as poisons. Adequate provision of information to the public, regular training of health care providers, better availability of drugs and antidotes and the establishment of poison information centres will facilitate in reducing the morbidity and mortality related to organophosphorus poisoning. Insecticides should be kept out of reach of children, to prevent accidental poisoning. During agricultural spraying, proper precautions should be taken to prevent inhalation and accidental ingestion of the substance.

OBJECTIVES

To analyze the patients with acute organophosphate poisoning with reference to

- ECG changes.
- Systemic manifestations.
- Relationship to the nature of compound.
- Outcome

MATERIALS & METHODS

Clinical parameters, CBC, Blood Sugar, Serum Urea, Creatinine, Serum NA+, Serum K+, LFT, ABG, ECG, Gastric aspirate analysis and Serum Cholinesterase.

BRIEF PROCEDURE

All patients admitted with ingestion of Organophosphorus compound was studied. The study was carried out over a 5 month period from June 2010. A detailed history was obtained and patients were subjected to thorough physical examination and ECG monitoring was done. Results of biochemical investigations including serum electrolytes, ECG was monitored.

INCLUSION CRITERIA

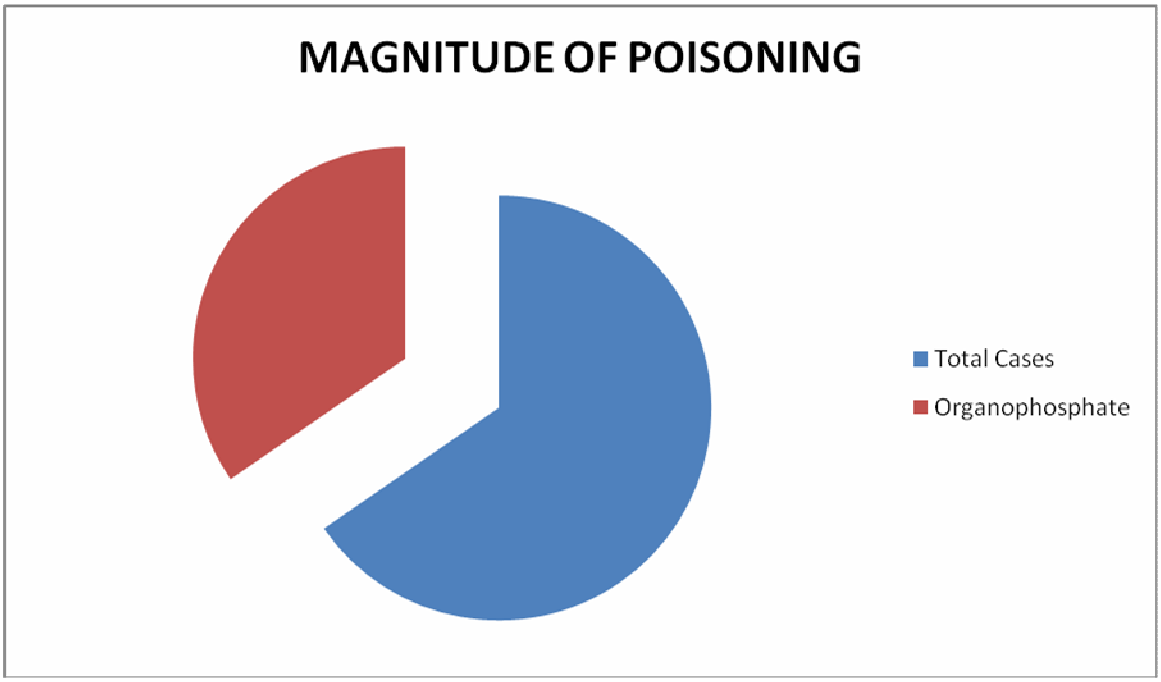
Patients admitted to the toxicology ward with ingestion of organophosporus compound poison.

EXCLUSION CRITERIA

1. Patients with history of cardiovascular disease.
2. Patients on Cardiac drugs
3. Patients with chronic renal failure.

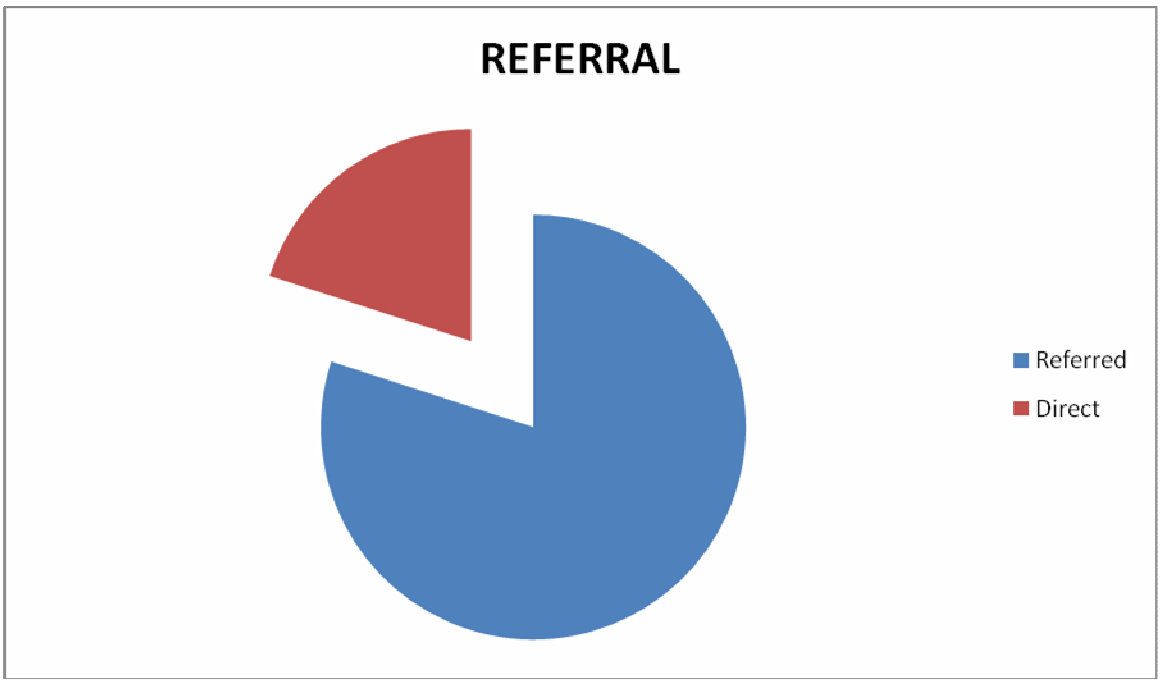
RESULTS

Epidemiological pattern of Organ phosphorus compound poisoning in poison centre, Government General Hospital.



Sl.No.	Cases	No of patients
1.	Total	255
2.	Organophosphate	135

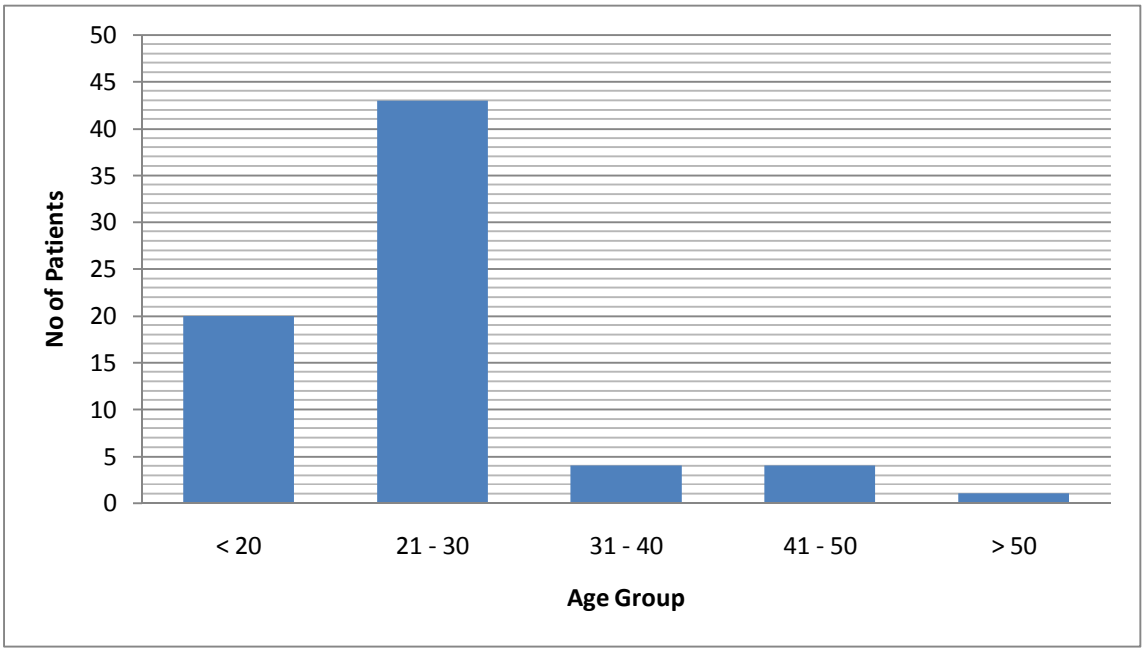
Organophosphate poisoning cases account for 53 % of the Total poison cases admitted.



Sl.No.	Referral	Frequency	Percentage
1.	Referred	108	80 %
2.	Direct	27	20 %

Majority of the patients of Organophosphate poisoning were Referrals

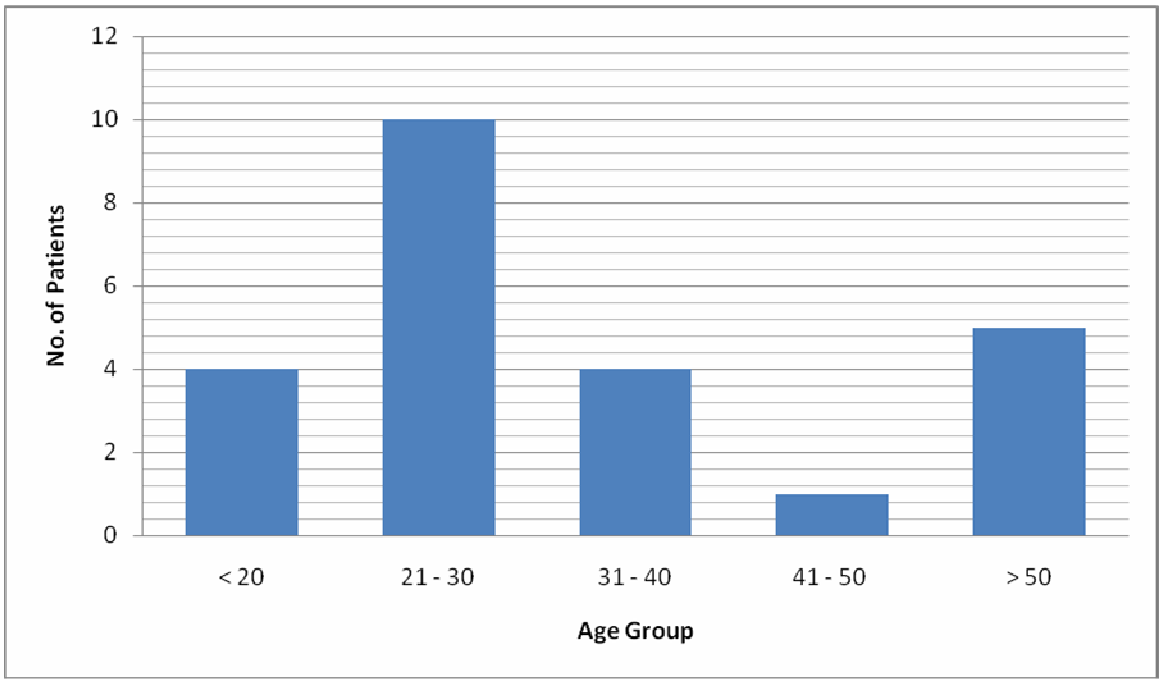
AGE PATTERN OF POISONING



Sl.No.	Age	Frequency	Percentage
1.	< 20	18	13.33 %
2.	21 – 30	59	43.70%
3.	31 – 40	26	19.25 %
4.	41 – 50	16	11.85 %
5.	> 50	16	11.85 %

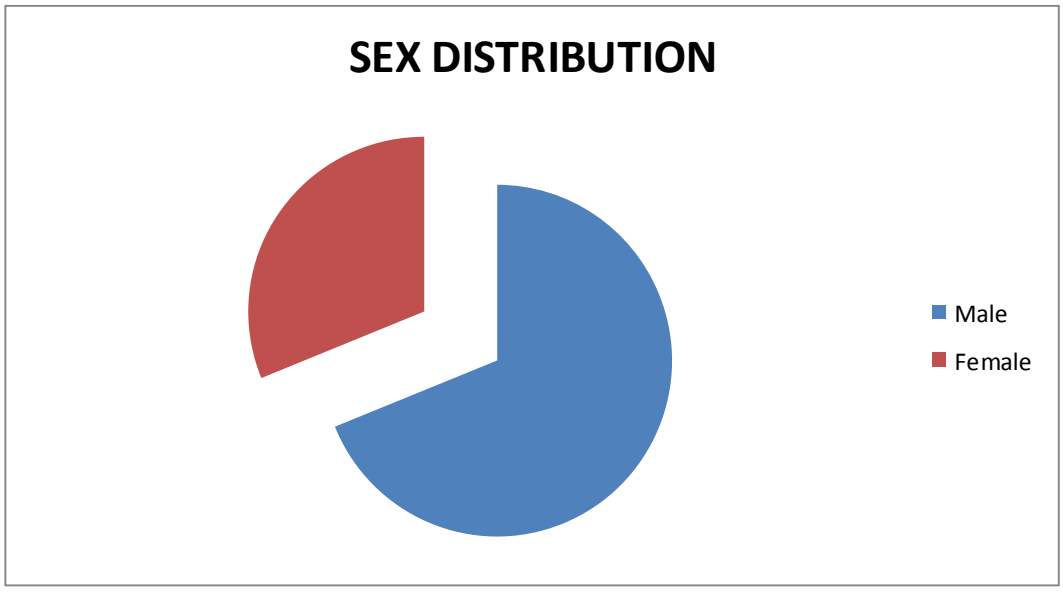
Majority of the patients are in the age group 21–30 Accounting for 59 %

AGE DISTRIBUTION AND MORTALITY



Sl.No.	Age	Frequency	Death	Percentage
1.	< 20	18	4	22.20 %
2.	21 – 30	59	10	16.94 %
3.	31 – 40	26	4	15 .38
4.	41 – 50	16	1	6.25 %
5.	> 50	16	5	31.125%

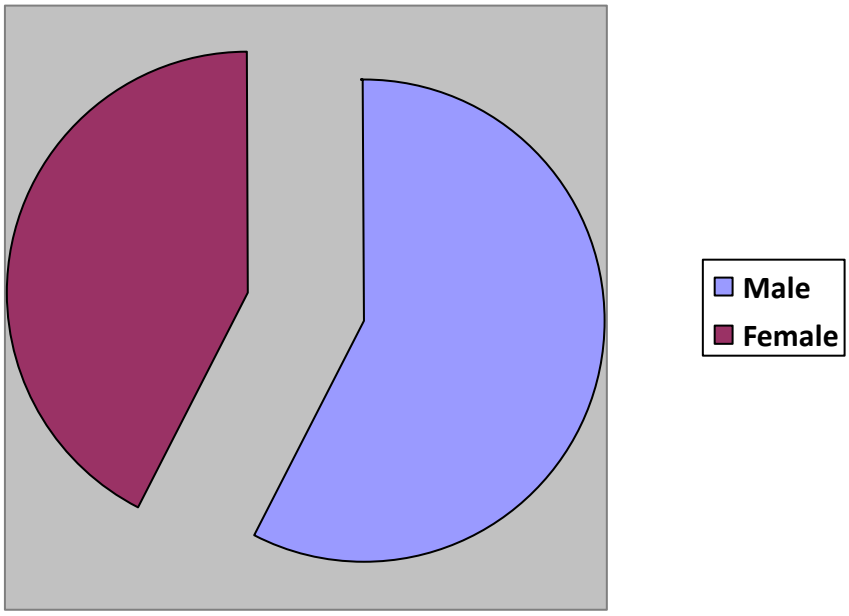
Percentage of death was noted to be increased in the extremes of age groups. 22.2% at <20 yrs and 31.12% at >50 yrs.



Sl.No.	Sex	Frequency	Percentage
1.	Male	95	70.37 %
2.	Female	43	31.85 %

Incidence of Organophosphate poisoning is more among Males.

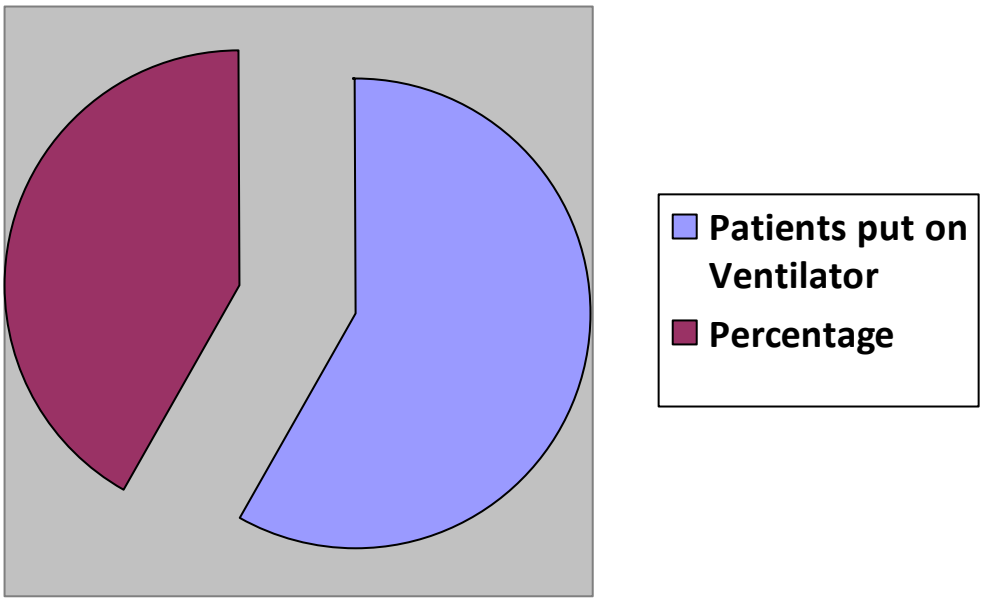
Mortality and Sex



Sl.No.	Sex	Frequency	Percentage
1.	Male	14	58.35%
2.	Female	10	41.6%

Mortality was more among males accounting for 58 .35% of the cases expired.

PATIENTS ON MECHANICAL VENTILATION.



Total Cases	-	135
Patients on Ventilator	-	35
Percentage of Patients put on Ventilator	-	25.32%

MORTALITY IN VENTILATED PATIENTS.

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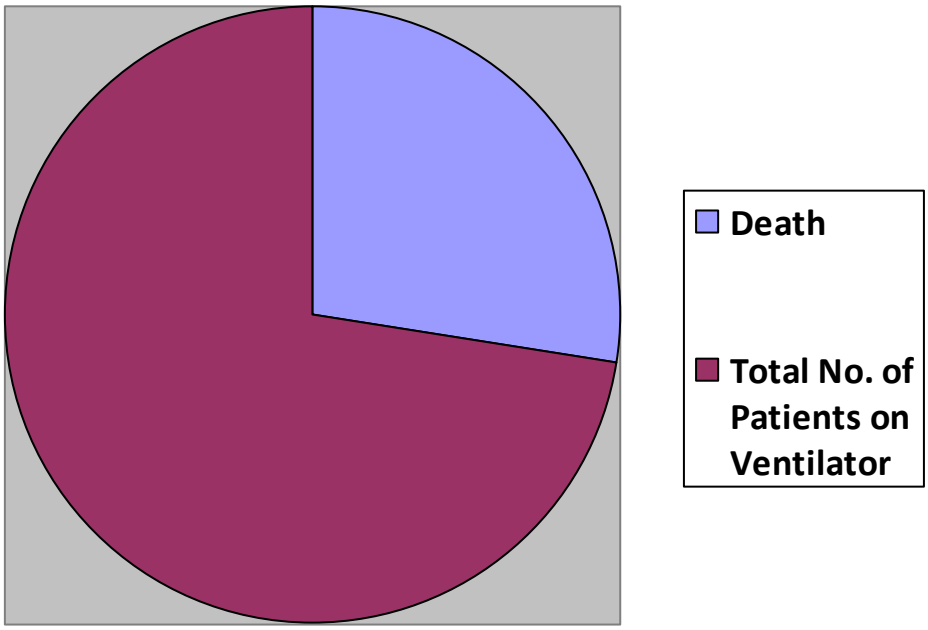


Total number of patients put on ventilator	–	35
Death	-	14
Percentage of Death in patients put on ventilator	-	37.14 %

COMPLICATIONS IN PATIENTS PUT ON VENTILATOR

S.NO.	COMPLICATION	NO. OF PATIENTS	PERCENTAGE
1.	Aspiration Pneumonitis	4	11.42 %
2.	Renal failure	4	11.42 %
3.	Cardiac Failure	3	8.57% %
4.	Sepsis	3	8.57 %
5.	HIE	2	5.71 %
6.	Respiratory failure	2	5.71 %
7.	Hepatic Failure	1	2.85 %
8.	Pneumothorax.	1	2.85 %

ANALYSIS OF CASES EXPIRED

Of the 24 Patients who had expired nature of the organophosphate was identified in 14 patients. Nature of the compound with regard to morbidity is as follows

S.No.	Compound	Duration of stay in Hospital	Complications	ECG Changes
1.	Acetate 15 % + Chlorpyriphos	4 Days		
2.	Monocrotophos	7 Days	Aspiration Pneumonitis	Tachycardia T Wave inversion
3.	Monocrotophos	8 Days	Hypothyroidism Aspiration Pneumonitis	QT Prolongation T wave inversion
4.	Monocrotophos	9 Days	HIE / Renal Failure	T wave inversion
5.	Baytex	4 Days	Respiratory Failure	
6.	Monocrotophos	15 Days		QT Prolongation
7.	Protenofos	19 Days	Sepsis	
8.	Quinofos	5 Days	Renal Failure	Tachycardia ST elevation
9.	Paraquat	13 Days	Liver Failure, Renal Failure	
10.	Monocrotophos	1 Day		QT Prolongation
11.	Methylparathion	2 Day		Tachycardia
12.	Dimethoat	1 Day	Hypotension	QT Prolongation

ANALYSIS OF CASES EXPIRED

Nature of the compound with regard to morbidity is as follows

S.No.	Compound	Duration of stay in Hospital	Complications	ECG Changes
13.	Unknown	1 Days	Hypotension	QT Prolongation
14.	Unknown	1 Days		
15.	Unknown	9 Days	ARDS, HIE, Sepsis	T wave inversion
16.	Unknown	3 Days	Post cardia arrest, HIE	Bradycardia
17.	Unknown	3 Days	Renal Failure, shock	QT Prolongation
18.	Unknown	3 Days	Respiratory failure, Renal Failure, Post cardia arrest and left pneumothorax	Bradycardia
19.	Methylparathion	3 Days	Acute renal failure	ST flattening
20.	Unknown	20 Minutes	Post cardia arrest, Metabolic encephalopathy	Tachycardia
21.	Unknown	5 Days		
22.	Monocrotophos	1 Day		QT Prolongation
23.	Unknown	2 Day		
24.	Unknown	1 Day		

Post Mortem changes

Post mortem was conducted for all the patients. Significant change was noted in one of the patients. The postmortem changes observed was

1. Heart was dilated.
2. Surface of the heart had petechial haemorrhage.
3. Thrombus was found in the coronary artery.
4. Chambers were filled with fluid blood.
5. Cardiac valves were normal.
6. The lungs were congested and filled with edematous fluid.

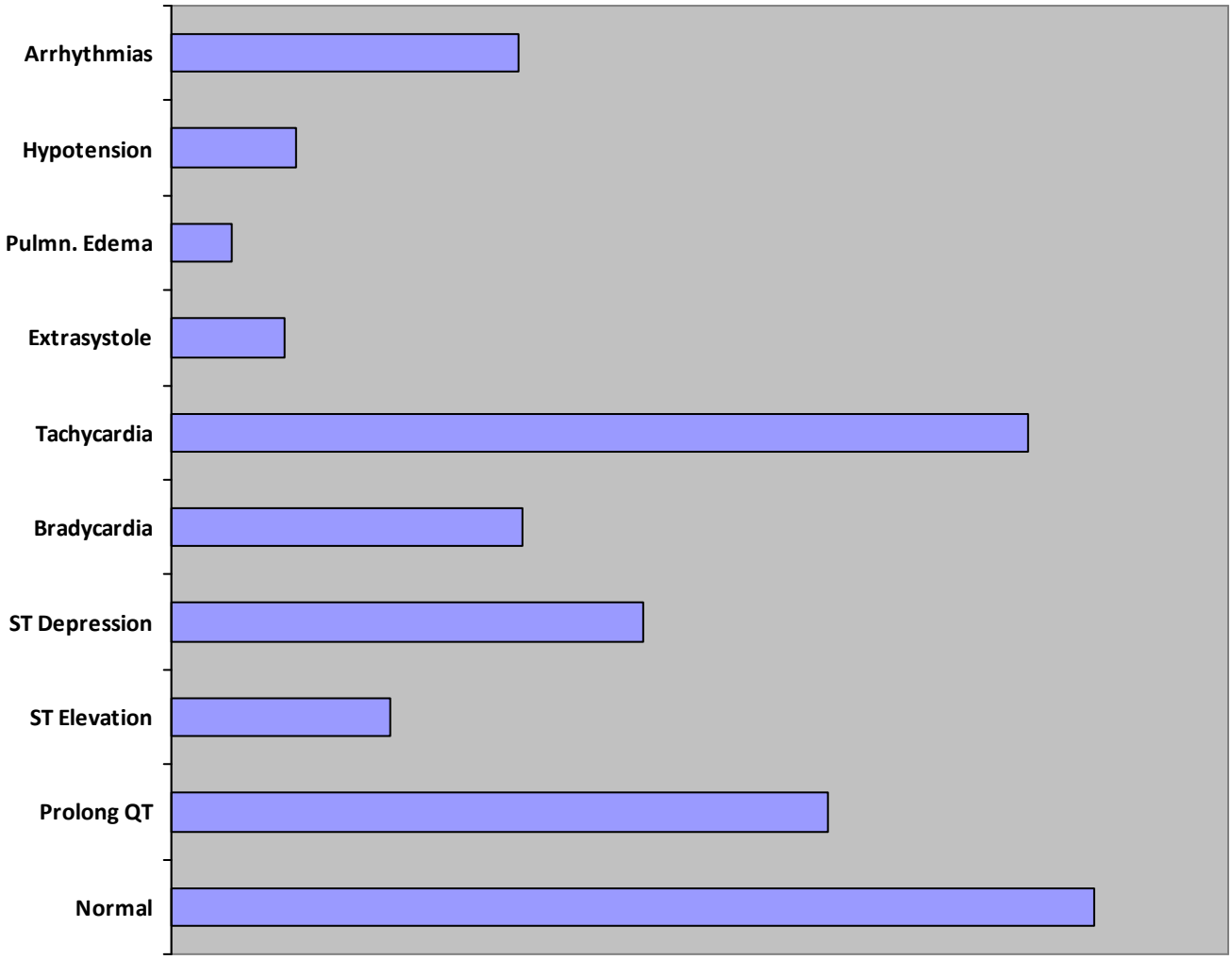
The post mortem changes in the other cases were

1. The heart was normal in size Two cases showed dilated heart.
2. Petechial haemorrhages was noted all over the surface of the heart
3. The cardiac valves and the coronaries were normal.
4. Atheromatous plaques was noted in the root of the aorta in a few cases.
5. The lungs were congested and filled with edematous fluid.

ECG AND CARDIAC CHANGES IN ORGANOPHOSPHATE POISONING

S.NO.	ECG CHANGES	FREQUENCY	PERCENTAGE
1.	Normal	59	43.7 %
2.	QT interval prolongation	41	31.1 %
3.	ST/T Wave Changes	35	26.1 %
4.	ST elevation	14	10.4 %
5.	T wave inversions	30	22.3 %
6.	Bradycardia	21	16.2 %
7.	Tachycardia	55	40.5 %
8.	Extrasystole	6	5.4 %
9.	Pulmonary edema	4	2.9 %
10.	Hypotension	8	5.9 %
11.	Arrhythmias	22	16.4 %

ECG AND CARDIAC CHANGES



No of patients:

In the observed ECG changes the maximum was Tachycardia followed by QT Prolongation.

BIOCHEMISTRY VALUES

1. RENAL FUNCTION TEST

S.NO.	VALUES	MEAN	SD (+/-)
1.	Blood Sugar mg/dl	114.20	74.30
2.	Urea mg/dl	26.94	10.20
3.	Creatinine	0.92	9.45
4.	Sodium	126.52	9.10

2. LIVER FUNCTION TESTS

S.NO.	VALUES	MEAN	SD (+/-)
1.	Bilirubin	0.81	2.30
2.	AST	24.24	20.32
3.	ALT	22.40	16.0
4.	Total Protein g/dl	6.5	0.5

Six patients showed features of renal failure. This was attributed to the hypotension following cardiac failure. One patient had elevated bilirubin with features of hepatic failure.

DISCUSSION

Comprehensive analysis of 135 cases of acute Organophosphate compound poisoning.

MAGNITUDE OF POISONING

In our Toxicology unit of Government General Hospital during the study period of 5 months, the Magnitude of the poisoning was 135 out of the total 335 cases that were admitted. This represents a total of 40.2 % of these cases 108 patient were referred from nearby Government and few private hospitals belonging to the five districts (Chennai, Kancheepuram, Thiruvallur, Vizhupuram and Vellore)

AGE PATTERN

In this series of 135 cases, the number of patients below 20 years was 13.33 %, between 21 – 30 = 43.70 %, 31 – 40 = 19.25%, 41 – 50 = 11.85 %, > 50 years = 11.85 %. The youngest was a patient aged 14 years and the oldest was a patient aged 85 years.

In a similar study by P. Karki JA Ansari etal of Singapore NA, the majority of patients was in 15 – 30 years = 65 % and the mean age group was 26.85 years. The series reported in this thesis had the similar pattern of age group affection. This is the most productive age groups.

The people in this age group are exposed to several pressures in the form of emotional conflicts social and economic pressures. This study stresses the importance of educative and preventive programs to be targeted in this age group.

SEX DISTRIBUTION

In our poison centre the distribution of Male was 95 (10.37%) and Female was 43 (30.31%). In a observation of 57 cases by P. Karki JA Ansari et al of Singapore, the percentage was 41 % for Male and 59 % for Female. The increased incidence of poisoning in our population could be because of easy accessibility to the poison by the Male. The majority of them were farmers.

OCCUPATION OF PATIENTS

The majority of the patients were farmers coming from near by villages.

DEMOGRAPHIC PROFILE

The majority of the patients were from the lower socioeconomic status being mostly farmers by occupation.

MAGNITUDE OF SUICIDAL POISONING

Of the 135 cases 9 were spray poisoning. The rest were suicidal in nature.

AGE PATTERN OF MORTALITY

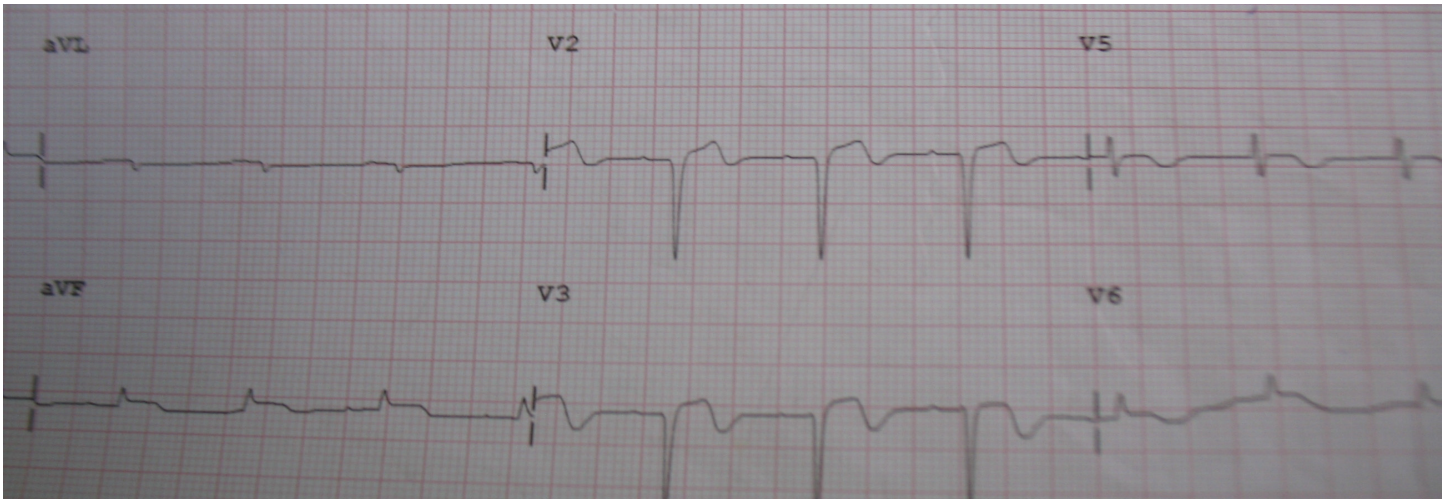
In our study the mortality among the different age groups were 20 = 2.96 %, 21 – 30 = 7.40 %, 31 – 40 = 2.96 %, 41 – 50 = 0.74 %, > 50 = 3.70 %.

The majority of the mortality was in the age group 21-30 years. The youngest patient was aged 14 years and the oldest aged 60 years. In a study by P. Karki JA Ansari et al, the major mortality was in the age group 15 – 30 years.

MORTALITY AND SEX

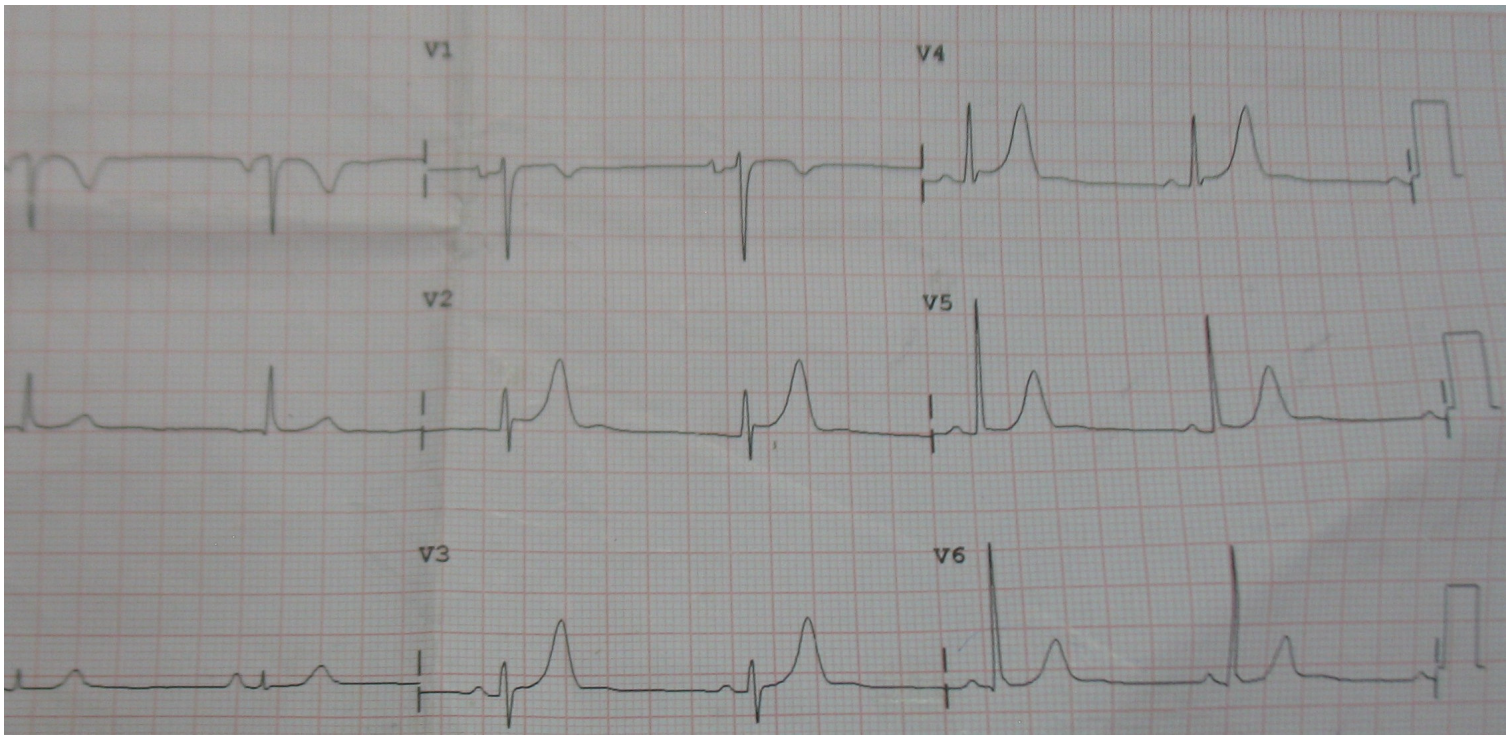
The percentage of mortality was higher in Males – 58 %, when compared to Females – 43.1 %. In a similar study by P. Karki JA Ansari et al, the mortality was more among Females.

ECG CHANGES

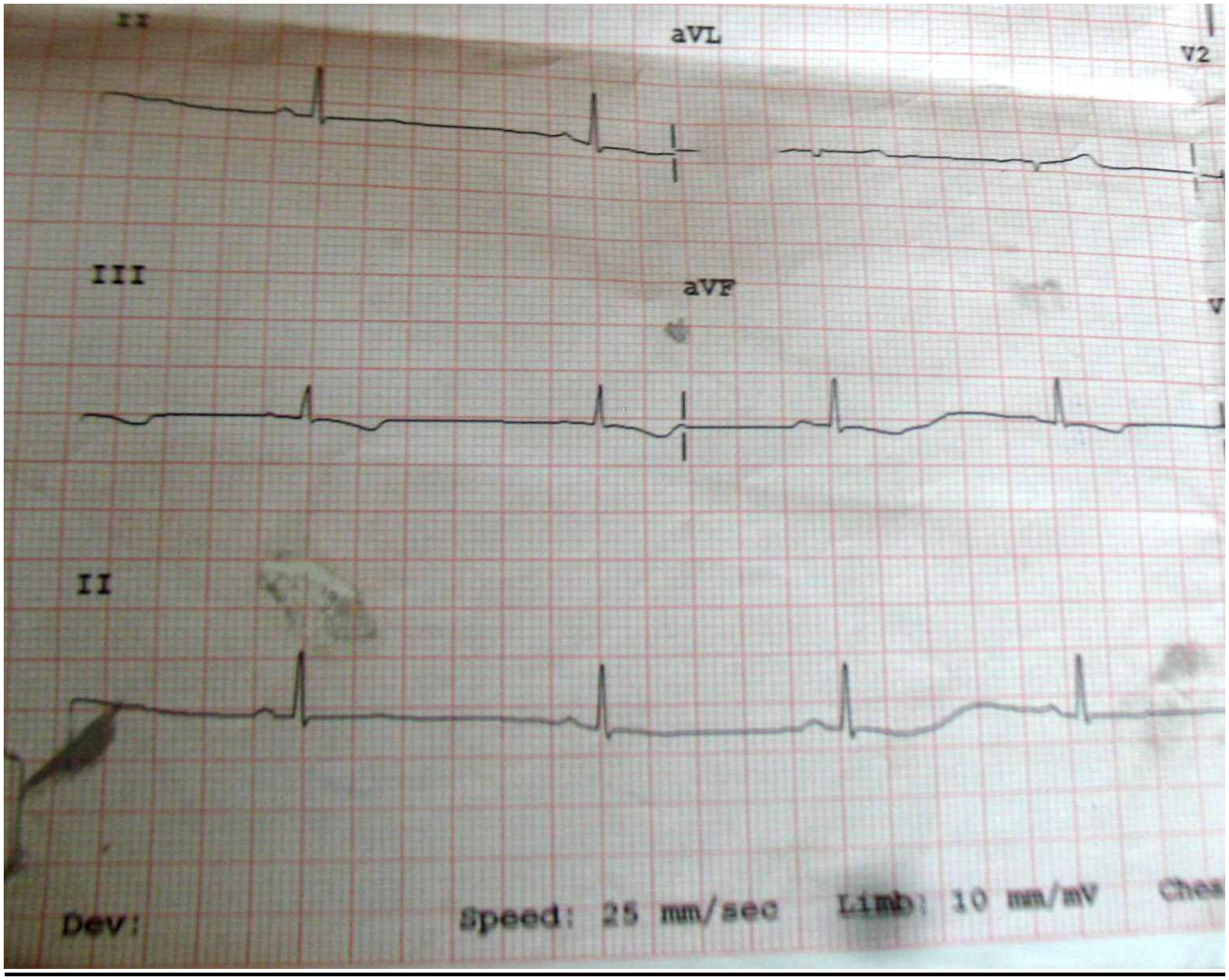


ECG showing ST Elevation in a patient with quinophos poisoning

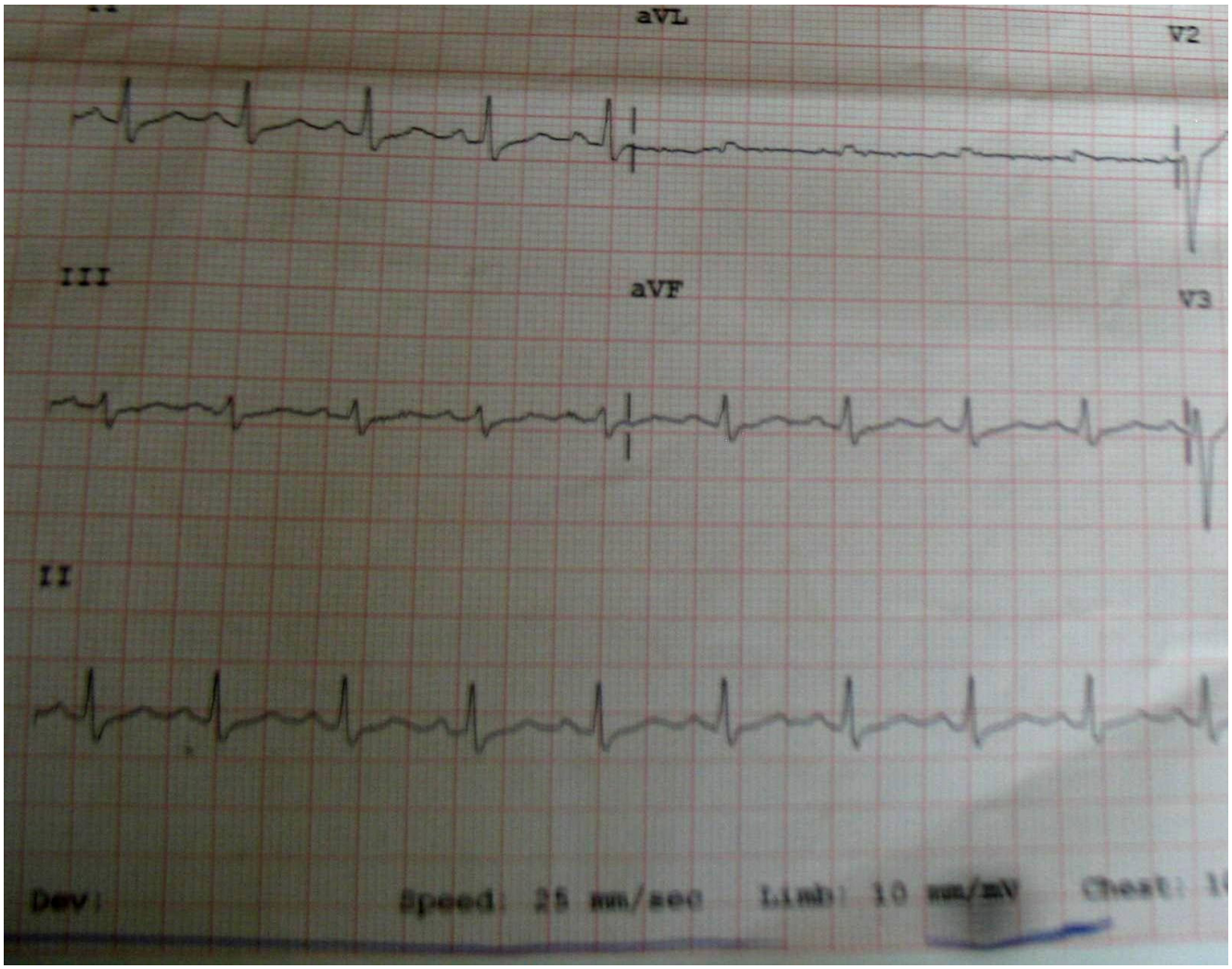
ECG showing Bradycardia in a patient with methyl parathionpoisoning'



ECG showing Bradycardia and Tall T Waves in a patient profeno phos poisoning



ECG showing Bradycardia and ST segment flattening in a patient with Monocrotophos poisoning



ECG showing QT prolongation in a patient with Monocrotophos poisoning

ECG ABNORMALITIES

Among the 135 patients significant ECG abnormalities were found in 56.3 %. 43.7 % had a normal rate and rhythm, Of the abnormal ECGs 41 (31.1 %) had QT prolongation 14 (10.4%) had ST elevations, 30 (22.3 %) had T wave inversions, 21 (16.2 %) had bradycardia, 55 (40.5 %) had tachycardia, 6 (5.4 %) had exrasystoles, 4 (2.9 %) developed pulmonary edema, 8 (15.9%) had hypotension, 22 (16.4 %) had arrhythmias.

A.M. Saadeh NA Farsakh et al observed cardiac manifestations in 67 %, ST changes 41 %, QT Prolongation 61 %, Bradycardia 28 %, Tachycardia 35 %, Pulmonary edema 43 %, arrhythmias 24 %, conduction defects 9 %, hypertension 22 % and hypotension 17 %.

ECG CHANGES IN CASES EXPIRED

15 of the 24 cases that had expired showed ECG changes accounting for 62.5 %. Of them QT prolongation was observed in 7 cases, Tachycardia in 4 cases, ST elevation in 1 patient, bardycardia in 2 patients and T wave inversion in 4 cases.

ECG CHANGES AND ITS RELATIONSHIP TO THE NATURE OF THE COMPOUND

ECG changes noted were mostly in the highly toxic compounds like monochrotophos, Quinophos, Chlorphyriphos, Baytex, Paraquate and Protenophos. Maximum manifestations were found in monochrotophos and Chlorphyriphos.

RENAL AND LIVER FUNCTION TESTS

Elevated renal parameters were observed in 7 % and elevated bilirubin and alterations in the liver functions was found inone patient.

RESPIRATORY COMPLICATIONS

Aspiration pneumonitis was observed in 4 patients. Pneumothorax was found in 1 patient.

MANAGEMENT

Of the 135 patients, except 2 all the others received stomach and body wash Atropine was administered to patients. The average of days of atropine requirement was 2days. P_2 AM infusion was administered to patients 112 patients. Ventilatory support was given to 35 patients.

MORTALITY

- The observed mortality was 17 %. Incidence of ECG abnormalities was 64 % in the mortality. The majority of the ECG changes were sinus tachycardia followed by QTprolongation. One patient had significant ST elevations indicating anterolateral myocardial infarct.

- 14 of the 24 patients who had died were put on ventilator indicating a percentage of 58.33 %

- Renal failure was observed in 4 patients 16.6 %.

- Liver failure was observed in one patient. He had multi organ dysfunction in the form of Renal failure, Respiratory failure and hypotension.

- Monocrotophos was he compound in which there was a maximum mortality.

The mortality in the series observed by P. Karki and JA Ansari et al was 8.1 %.

The mortality in the series observed by AM Saadeh NA Farsakh et al was 4 %

The mortality in our population is higher. This could be attributed to the fact the ours is a referral centre, receiving seriously ill patients from nearby hospitals.

The case fatality of self poisoning in the developing countries in 10–20 %
In the western countries, the case fatality is 0.3 % [14]

CONCLUSION

1. Organophosphorus compound is the most widely used suicidal agent in our population.

2. The incidence is highest among farmers.

3. Majority of the poisoning are suicidal. Few are accidental due to spray poisoning.

4. In our observation, incidence is higher in Males than in Females.

5. The highest incidence is found in the age group of 20 – 30 years

6. Death is affected by the nature of the compound consumed. The compounds with highest mortality were monochrotophos and Chlorphyrifos.

7. ECG abnormalities are found in the majority of the patients, accounting for a percentage of 56.3 %. The major ECG abnormality noted was Tachycardia followed by QT prolongation, bradycardia, ST elevations. The mortality was higher in patients with significant ECG changes. The incidence of ECG abnormalities was higher in the mortality group accounting for 64 %.

8. Prognosis is poor in patients presenting with respiratory failure.

STRENGTHS AND LIMITATIONS

1. The majority of the patients were referrals who would have a higher morbidity and mortality.
2. It is a single centre study.
3. Children are not represented.

RECOMMENDATIONS

Organophosphate compound poisoning and mortality is a huge burden on the productive population. It is easily available over the counter. Monitoring the sale of these compounds may help to limit its availability as a suicidal agent. Steps to educate the farmers and provide counseling can be undertaken.

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"Cardiac Manifestations of Organo Phosphates as monitored by ECG as a Predictor Of Morbidity And Mortality In Patients With OPC Poisoning"

PROFORMA

GENERAL:

NAME: AGE: GENDER:
MRD NO: UNIT:
DOA: TIME:

BRIEF PRESENT ILLNESS:

Date and time of poisoning :
Time elapsed since ingestion of poison :
Nature of compound :

PAST HISTORY:

[ ] DM Duration:_____
[ ] HT Duration:_____
History of previous poisoning :
Other relevant past history:

PERSONAL HISTORY:

[ ] Smoking Duration:_____ Quantity:_____
[ ] Alcoholism Duration:_____ Quantity:_____

PHYSICAL EXAMINATION:

GENERAL EXAMINATION:

MEASUREMENTS:

Height :……. cm
Weight :……. Kg
BMI :……. kg/m^2

VITALS:

PR: / min
[ ] Bradycardia {<60}
[ ] Tachycardia {>100}

TEMP:
[ ] < 98^0 F
[ ] 98-99^0 F
[ ] > 99^0 F

RR: / min
[ ] < 20
[ ] 20 – 30
[ ] > 30

BP: / mmHg
[ ] Hypotension {<90/60}
[ ] Normotension {90-120/60-80}

SYSTEMIC EXAMINATION:

CVS:

RS:

ABDOMEN:

CNS:

INVESTIGATIONS

INVESTIGATIONS UNDER STUDY:

ECG - Rate
Rhythem
Axis
P Wave
PR interval
QRS duration
ST segment
T Wave
U Wave
Comments:

ECHO

CPK - MB

OTHER INVESTIGATIONS:

TC	:
DC	:
ESR (1hr)	:
HB	:
PCV	:
Platelets	:
Blood Urea	:
Sr. Creatinine	:
Sr. Sodium	:
Sr. Potassium	:
Total Bilirubin	:
AST	:
ALT	:
SAP	:
Total Protein	:
Total Cholesterol	:
TGL	:
LDL	:
CPK	:
CPK MB	:
Trop T	:
Trop I	:

:

IMAGING

CHEST XRAY :

ECHO :

TREATMENT

		OTHER REMARKS
Day 1		
Day 2		
Day 3		
Day 4		
Day 5		
Day 6		
Day 7		

FOLLOW UP